Table
Demographics and outcomes of patients a BSI after SCT. 13 patients had >1 infection accounting for 39 total infections.

	MBI-LCBI (n=18)	CLABSI (n=20)	Secondary BSI (n=13)	> 1 BSI after HSCT (n=13 patients)
Mean age (years) at SCT (Range)	7.1 (0.9-28.7)	4.6 (0.3-29.8)	5.4 (0.9-32.8)	9.2 (2.2-27.5)
Median days from SCT to BSI (IQR)	7 (4-55)	59 (8-137)	70 (48-118)	149 (58-313)
Diagnosis				
Immune Deficiency (n=32)	8 (44%)	13 (65%)	4 (31%)	7 (54%)
Malignancy (n=17)	6 (33%)	5 (25%)	3 (23%)	3 (23%)
Marrow Failure (n=8)	2 (11%)	0	3 (23%)	3 (23%)
Benign Hematology (n=5)	1 (6%)	1 (5%)	3 (23%)	0
Genetic/Metabolic (n=2)	1(6%)	1 (5%)	0	0
Myeloablative conditioning (n=28)	8 (44%)	12 (60%)	5 (38%)	3 (23%)
GVHD (Grade II-IV) at 100 days (n=19)	4 (22%)	5 (25%)	6 (46%)	4 (31%)
Sepsis/shock (n=31)	4 (22%)	10 (50%)	3 (23%)	14 (36%)
ICU care within 7 days of BSI (n=15)	4 (22%)	4 (20%)	3 (23%)	4 (10%)
Died (n=27)	8 (44%)	8 (40%)	3 (23%)	8 (62%)
Non relapsed mortality (n=20)	6 (33%)	6 (30%)	3 (23%)	5 (38%)
Median days from BSI to death (IQR)	90 (51-152)	119 (50-206)	44 (34-63)	
In patients with > 1 BSI, median days from last BSI to death (IQR)				156 (113-302)

Methods: We reviewed all BSIs in SCT patients from May 2011 to April 2014 at our center to determine the timing, underlying diagnosis, and outcomes of patients diagnosed with MBI-LCBIs, CLABSIs and secondary BSIs after stem cell transplant (SCT). We applied the new MBI-LCBI classification to patients undergoing SCT prior to NHSN criteria implementation.
Results: 299 patients underwent SCT during the 36-month study period. 34 CLABSIs, 30 MBI-LCBIs, and 26 secondary BSIs were diagnosed in 64 patients (21%). Thirteen patients (20%) had more than one infection accounting for 39 of the 90 (43%) infections. MBI-LCBIs occurred shortly after SCT at a median of 7 days after transplant. 11 of 14 patients with MBI-LCBIs after day +30 (including patients with > 1 infection) were diagnosed with GVHD, with 10/11 having GI-GVHD.
Conclusions: One third of BSIs were classified as an MBI-LCBI. MBI-LCBIs occurred earlier after SCT than CLABSIs and secondary BSIs, likely due to mucositis, and were associated with GVHD after day +30. As the MBI-LCBI classification is new, further research is needed to understand the pathogenesis and prevention of MBI-LCBIs.

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Pulmonary Hypertension Associated with Bronchiolitis Obliterans after Stem Cell Transplant

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Background: Bronchiolitis obliterans (BO) and pulmonary hypertension (PH) are rare and fatal complications of stem cell transplantation (SCT). BO is a non-reversible obstructive lung disease in which bronchioles are compressed and narrowed by fibrosis and/or inflammation. PH arises from increased pulmonary vascular resistance leading to increased right ventricular pressure, right heart failure, and death. PH has not been described in patients with BO after SCT.
Design/Methods: To evaluate PH in patients with BO, we retrospectively reviewed all cases of BO in 291 patients undergoing allogeneic HSCT from January 2009 to December of 2012. Patients diagnosed with BO met standard criteria for BO diagnosis including high resolution CT evidence of air-trapping. PH was diagnosed with echocardiography, cardiac catheterization, and autopsy.
Results: Four patients (1.4%) were diagnosed with BO. All four patients had a history of graft versus host disease, no infectious cause of respiratory symptoms, and high resolution CT evidence of BO. Two patients received PFT testing which showed decreased FEV_1. All patients received serial echocardiography after SCT. Three of the four patients were diagnosed with PH after the diagnosis of BO **(Table)**. All three patients had elevated estimated right ventricular pressure, with secondary signs of PH, on echocardiography. One patient underwent cardiac catheterization confirming PH, and one had findings consistent with PH on autopsy **(Figure)**. Two patients died from respiratory failure, both received inhaled nitric oxide. The surviving patient with PH and BO required intermittent non-invasive ventilation and was treated with sildenafil for PH. The patient without PH died 192 days after SCT.
Conclusion: PH has not been described in patients with BO after SCT. The etiology of PH after BO is unclear; however, parenchymal changes in BO may lead to pulmonary vascular injury and hypertrophy. Also, long-term hypoxemia and inflammation can lead to vasoconstriction, vascular remodeling and angiogenesis, which in turn leads to increased

Table
Patients diagnosed with BO and PH after HSCT

Diagnosis	Age at SCT (years)	Days from SCT to diagnosis of BO	Days from SCT to diagnosis of PH	Days from SCT to death
WiskottAldrich	0.6	233	235	Alive
AML	17.4	305	351	352
CML	10.4	158	247	1368
Aplastic Anemia	5.4	131	No diagnosis of PH	192

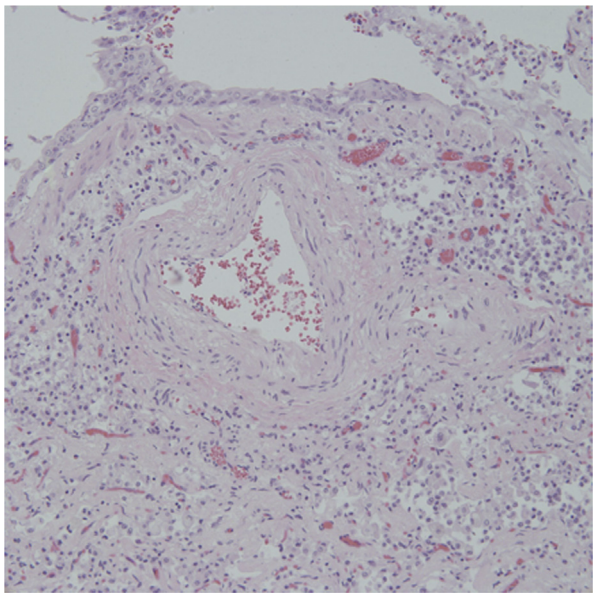

Figure. Pulmonary arteriole intimal and medial thickening seen in a patient with bronchiolitis obliterans

vessel wall proliferation. Routine screening for PH in patients with BO may assist in diagnosing PH. The treatment for PH differs from BO and there is little understanding of their interplay post-HSCT; further investigation is needed.

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CT Scan Frequently Misses the Diagnosis of Posterior Reversible Encephalopathy Syndrome (PRES) after Stem Cell Transplant

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Background: Posterior reversible encephalopathy syndrome (PRES) is a clinical syndrome characterized by vision changes, altered mental status, and seizures that are typically caused by acute rise in blood pressure and resolve over time. PRES has been reported after stem cell transplant (SCT) in association with hypertension from calcineurin inhibitors and steroids. The radiologic evaluation of PRES after SCT has not been well described.
Design/Methods: A retrospective review of all cases of PRES in SCT recipients from January of 2004 to December of 2013 was performed. PRES was diagnosed if the patient developed encephalopathy, headache, seizures or visual disturbances with a CT and/or MRI showing imaging findings compatible with PRES.
Results: Twenty-two of 838 (2.6%) transplanted patients were diagnosed with PRES, all of them allogeneic SCT recipients. The majority (64%) were male with a median age of 9.4 years (IQR 4.9-12.1) at time of SCT. Nine patients had a marrow failure syndrome (41%), 7 of which had Fanconi Anemia; 7 patients had an immune deficiency (32%); 5 had an underlying malignancy (23%); and 1 patient had a metabolic syndrome (4%). Twelve patients (55%) had a myeloablative preparative regimen. All patients received cyclosporine for graft versus host disease prophylaxis after SCT and 11 (50%) were treated with additional immunosupressants including steroids for GVHD at the time of event. PRES was diagnosed at a median of 49 days (IQR 31-88) after SCT. Nineteen patients (86%) presented with seizures, the other 3 (14%) had altered mental status. All patients underwent a brain CT and/or MRI, 21 of 22 patients (95%) received a CT scan when they became symptomatic, which was diagnostic of PRES in 8 of the 21 studies (38%). Eighteen patients (82%) received MRI, 17 of the 18 (96%) were consistent with PRES. The one MRI not consistent with PRES was done 20 days after initial diagnosis, subsequent to resolution of the abnormal CT findings. Notably in 13 patients initial CT scans did not demonstrate findings of PRES, which were subsequently found on MRI. The median time elapsed between CT and an MRI examination was 20 hours (range: 3.6 hours to 9 days). The overall survival in patients that developed PRES was 35% (8 of 23 survived).
Conclusion: CT scan serves as a good diagnostic test in emergency situations to rule out CNS bleed or infection in SCT patients with acute mental status changes or seizures, but it is not an adequate radiologic study for diagnosing of PRES after SCT. Patients with clinical symptoms suggestive of PRES, but negative CT, should undergo MRI of the brain after the acute event is controlled to assist in diagnosis. MRI results diagnostic of PRES would prompt physicians to provide good hypertension control and aid decision-making regarding invasive diagnostic procedures (like lumbar puncture) or intensification of empiric antimicrobial therapy.

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A Pilot Study of Donor Enteral Human Milk to Modulate the Gut Microbiome in Children Receiving Stem Cell Transplant

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Background: The gut microbiome is an important immune modulator, and previous work has shown important changes in the gut microbiome associated with the occurrence of GVHD. Studies in premature babies at risk for necrotizing enterocolitis have shown that enteral human milk reduces gut inflammation and bacterial translocation (Morrow et al, 2012). We hypothesized that enteral human milk given in the peri-transplant period would be well tolerated and would modify the microbiome and reduce inflammation.
Methods: In a pilot study, we treated 10 children <2 yrs with donor enteral milk, and collected stool samples for microbiome analysis and blood samples for analysis of inflammatory markers. Milk was obtained from the Mother's Milk Bank of the Northeast. Milk donors underwent standard donor screening and milk was pasteurized before use. As prebiotic and anti-inflammatory benefits of are thought to derive from human milk oligosaccharides produced by *fucosyltransferase2* (FUT2) and related gene biosynthesis